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Environmental Research 198 (2021) 111189

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

Simple quantitative assessment of the outdoor versus indoor airborne transmission of viruses and COVID-19

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ARTICLE INFO

Keywords:
COVID-19
Virus airborne transmission
Outdoor versus indoor transmission
Quantitative risk assessment
Meteorological and topographical influence

ABSTRACT

In this paper we develop a simple model of the inhaled flow rate of aerosol particles of respiratory origin *i.e.* that have been exhaled by other people. A connection is made between the exposure dose and the probability of developing an airborne disease. This allows a simple assessment of the outdoor versus indoor risk of contamination to be made in a variety of meteorological situations. It is shown quantitatively that for most cases, the outdoor risk is orders of magnitude less than the indoor risk and that it can become comparable only for extremely specific meteorological and topographical situations. It sheds light on various observations of COVID-19 spreading in mountain valleys with temperature inversions while at the same time other areas are much less impacted.

1. Introduction

The purpose of the present paper is to develop a simple quantitative assessment of the relative risk between indoor and outdoor environments for the so-called "aerosol" or "airborne" transmission of viruses and for different outdoor situations. The goal is to assist in public health policy and recommendations.

Respiratory diseases represent a serious burden for global public health. It should be remembered that in western countries prior to the advent of antibiotic drugs, they were the primary causes of death. Antibiotics, however, are essentially inactive for virus borne illnesses, except in their ability to prevent secondary infection. In the case of mutating viruses, vaccines have to evolve constantly as in the case of influenza viruses. Therefore, it is essential to understand the problem of viral transmission in order to provide effective guidance for the mitigation of epidemics.

Amongst respiratory diseases and according to the World Health Organization, influenza which is caused by viruses of various kinds, leads to the premature death each year of between 290,000 and 650,000 people (WHO, 2017) and measles more than 140,000 (WHO, 2019b). At the beginning of the 21st century, new respiratory viruses have appeared such as the SARS-COV-1 in 2003, a coronavirus that emerged first in China in 2002 and caused severe respiratory disease often leading to pneumonia with a rather high (around 10%) mortality. Fortunately, the spread of that epidemic was limited mainly and came to an end before the end of 2003 (CDC, 2017). Therefore, despite its seriousness, the total mortality of SARS-COV-1 remained low. More recently in 2009, the H1N1 pdm2009 flu virus emerged, and it has been estimated by the CDC (US Center of Disease Control), that during its first year of circulation, it killed 0.001–0.007% of the world population (CDC, 2020). It has been circulating since, causing significant health problems in various countries.

For comparison, previous epidemics in the last century included the 1968 H3N2 flu which killed around 0.03% of the world population (CDC, 2019b), and the 1918 H1N1 pandemic (the so-called Spanish flu) had a much more terrible impact ranging from 1 to 3% (CDC, 2019a).

The current pandemic linked to the new SARS-COV2 coronavirus which emerged in China at the end of 2019, has already resulted in mortality close to 0.036% of the world population (JHU, 2021). The illness caused by this virus has been named COVID-19 for COronaVIrus Disease of 2019 and has led many governments to take stiff measures such as lockdowns, with severe damage inflicted on the economy and secondary effects on health. Therefore, and as stated above, good knowledge of the actual transmission routes is essential in order to take rational and scientifically based decisions to mitigate virus spread without destroying social life and the economy.

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https://doi.org/10.1016/j.envres.2021.111189
Received 5 January 2021; Received in revised form 6 April 2021; Accepted 12 April 2021
Available online 16 April 2021

It is commonly admitted that respiratory viruses are transmitted in three ways. The first is via "direct contact": it means that an infected person can transmit a given amount of virus to a person in close contact, either by sneezing or coughing and even talking and breathing, thus emitting a variety of micro-droplets that can be projected directly onto the mucosa (lips, nose and eyes) of the receiving person, or onto the skin and clothes, and subsequently transmitted by the hands to the mucosa. The second way is linked to objects that have been contaminated in the same way and referred to in medical science as "fomites": it is then expected that, even without direct contact with the infected person, touching the contaminated object with bare hands can lead to contamination. The third route involves a persistent aerosol formed by the smallest particles emitted by an infected person that can subsequently be breathed in. This is known as "airborne transmission" or "aerosol transmission".

At the beginning of the COVID-19 pandemic, airborne transmission was minimized, if not outright denied, by health authorities either by the WHO or by governmental agencies in a variety of countries, such as the CDC (Center of Disease Control) in the US or the HAS in France (Haute Autorité de Santé). Therefore, the recommendations for mitigation of the epidemic were mainly based on the first two methods of transmission: social distancing which means not coming into close contact with someone else (with a recommended distance between 1 and 2 m as defined in different countries), together with frequent washing and disinfecting hands and surfaces. Finally, in addition to these preventative measures for individuals, a mitigation strategy of testing-tracing-isolating that may harm privacy and promote digital surveillance, based on new digital tools and tests, was often adopted (Rowe et al., 2020).

However, several reasons, based as much on scientific work as on observation, cast great doubt on the fact that aerosol transmission could be negligible. In fact, it appears now that it could be a major way of transmission in addition to close contact. The key role for fomites itself is now contested (Goldman, 2020).

Microdroplets that move in the air experience a drag force F_D that results in a terminal velocity. For small particles, the drag force follows the well-known Stokes law (Stokes, 1851) and is proportional to the radius of the particle:

$$F_D = 6\pi\mu r v \tag{1}$$

where μ is the air viscosity, with r and v being the radius and velocity of the particle respectively.

The force of gravity experienced by the particle is proportional to its mass *i.e.* to the third power of the radius, hence it is understandable that, below a given size, particles can remain in air (as fog) for a very long time and even nearly indefinitely, due to the natural and relentless movement of the atmosphere, whether indoors or outdoors.

For a relative humidity below 100%, the microdroplet can evaporate in a very short time (see Supplementary Materials, hereafter SM), with a reduction in volume of an order of magnitude or more (Morawska, 2006; Nicas et al., 2005; Vejerano and Marr, 2018) due to water loss. This can lead to the formation of dry nuclei with a high biological load including viruses. Due to their size reduction, these particles will remain airborne and represent a serious infectious hazard.

These phenomena were recognized and developed in a visionary paper by Wells as early as 1934, which concluded that they result in contaminated air which can lead to the contagion of persons just by respiration (Wells, 1934). This third route of contamination is well recognized in a variety of diseases such as measles (WHO, 2019a).

Due to the importance of respiratory diseases, researchers did not wait for COVID-19 to study and characterize aerosols emitted by human beings (Morawska et al., 2009) either by simple respiration or by coughing, sneezing, talking, and singing. The behavior of the emitted aerosols (Bourouiba et al., 2014; Bourouiba, 2020) and the problem of their infectiousness (Buonanno et al., 2020), has also been studied. In fact, the scientific community has been the first to raise a cry of alarm regarding the most probable role of aerosol transmission (Borsellino et al., 2020; Morawska and Cao, 2020; Morawska and Milton, 2020).

It is mainly observations however, that have led to the conclusion that airborne transmission is a key contamination route for COVID-19. Several situations have been reported involving high contamination rates at a given location, known as "super-spreader" or "cluster" events. An exceptionally large majority of these are indoor events which, as we shall see, is consistent with airborne transmission. These include contamination on cruise ships (Azimi et al., 2021), public transportation (Yang et al., 2020), restaurants (Lu and Yang, 2020), religious ceremonies (James et al., 2020) amongst others. The key role of aerosol transmission in these events has been discussed in a recent paper (Shen et al., 2020).

The seasonality of influenza is well known (Lofgren et al., 2007; Tamerius et al., 2013) and it is now largely admitted in the case of COVID-19 (Mattiuzzi et al., 2021). The importance of atmospheric parameters such as temperature and humidity are also well recognized in both cases (Marr et al., 2019; Pica and Bouvier, 2012). The strong correlation with weather and climate could be partly due to physiological reasons (Eccles, 2002; Rahman and Williams, 2021) but is a strong argument for the aerosol route.

Other arguments could come from other observations: the occurrence of straightforward pneumonia in some patients without any symptoms in the upper respiratory tract is completely consistent with the inhalation of infective microdroplets directly into the lung (Karimzadeh et al., 2021; Yezli and Otter, 2011). A difference has also been observed between the occurrence of severe forms of the disease between males and females (Peckham et al., 2020), with fatal outcomes being more probable for men. Apart from other physiological reasons, which are beyond the scope of this paper, we suggest that the difference between male and female respiration, males having a much deeper inspiration (LoMauro and Aliverti, 2018) together with aerosol transmission, could at least partly explain this difference.

In the present paper, simple calculations and arguments lead to a remarkably simple formula for quantifying the relative level and dose of exposure to the disease (which will be defined in section 3) and the relative probability of developing a disease between indoor and outdoor situations. Of course outdoor meteorological parameters are essential for quantitative assessments, a problem closely related to the science of air pollution.

Outdoors, only special situations of temperature inversion could lead to situations with an outdoor risk comparable to that found indoors. The discussion sheds light on the climatic and weather correlations that have been observed with the spread of the disease.

The present paper is organized as follow: in section 2 a short review is given of what is known about human exhaled droplets and aerosols. In section 3, modeling indoor situations is then discussed and a simple calculation of the level and dose of exposure is presented (together with their definitions). Note that the extensive current knowledge of human aerosols briefly reviewed in section 2 is not needed nor used for the sake of simplicity in section 3. In section 4 an airshed calculation of the outdoor level of exposure is described. Here the main unknown is the airshed "height" which is discussed at length in the SM. The respective relative level of exposure and of disease probability between outdoor and indoor situations are discussed in section 5 with numerical applications and a general discussion. Section 6 deals with atmospheric markers of outdoor and indoor risks.

The main result of the present work, highlighted in section 5 and in the conclusion (section 7) is that the outdoor risk, except in special meteorological situations with a very stable atmosphere and exceptionally low wind, is generally far lower than the indoor risk (often by orders of magnitudes) and that indoors, **the fresh air ventilation rate is a key factor in mitigating this risk.** This last point has been pointed out by several researchers (Gao et al., 2016; Morawska et al., 2020) and is now recognized by the authorities. What is really new in our

contribution is the application of the "rebreathed air" concept to outdoor situations and the use of relative risk and probability between two kinds of exposure.

2. Human emissions

2.1. Human respiratory characteristics

The first feature of human respiration is that we inhale fresh air mainly composed of nitrogen (78%) and oxygen (21%) and of various minor species including carbon dioxide (0.04%), natural aerosols and possible pollutants. In the exhaled air, the concentration of carbon dioxide is enhanced to a much larger value, typically about 4–5% (Nerovnov et al., 2017). It also contains a variety of microdroplets of various sizes that come from the respiratory tract and are mainly composed of water (98.2%) (Chen and Zhao, 2010). As discussed in the introduction and in the SM, the largest microdroplets fall to the ground over a relatively short distance. Hence the recommendation of social distancing of 1 to 2 m. However, the smallest particles are able to stay in suspension in the air leading to the creation of a "human" aerosol. It has been commonly admitted that the dividing line between the two cases is defined for a radius of 5 μm (Gralton et al., 2011) although such a simple discrimination has been largely disputed and that a variety of "dividing lines" can be found in the literature (Bourouiba et al., 2014; Bourouiba, 2020; Morawska, 2006). The mean value at rest of the exhaled (inhaled) volume for an adult, is 500 ml (Tortora and Derrickson, 2016) with a normal frequency of 9–12 cycles per minute (Barrett et al., 2012) which leads to a mean air flow rate of around 5–6 l/min that is used in the present work. Note that the frequency hardly changes with greater physical activity and the higher flow rate comes from a higher inhaled/exhaled volume.

These water-based microdroplets contain mucus and possibly viruses and bacteria, hence their potential role in contamination. This fact has led various researchers to study the physical and biological characteristics of emitted microdroplets. Clearly a study of their size distribution is fundamental to know if their aerosolization is possible, leading then to this route of contamination by an infected person.

As far back as 1945, Duguid conducted experiments by collecting exhaled microdroplets on celluloid slides followed by micrometry. His results were published in a seminal paper (Duguid, 1945), however in those days, most of the very powerful modern in-situ particle size analyzing methods did not exist. Since then, numerous studies have been performed (see for example (Alsved et al., 2020; Yang et al., 2007). Amongst them, the most advanced facility dedicated to this problem has been built at the Queensland University of Technology, in Brisbane, Australia. A special wind tunnel (Morawska et al., 2009) allows a human emitter to be isolated in completely clean air (*i.e.*, aerosol particles in the air are removed prior to the experiment) and a variety of particle size analyzers are used to derive the complete particle size distribution. The research team led by L. Morawska has published several papers (Johnson and Morawska, 2009; Johnson et al., 2011; Morawska et al., 2009) amongst others which provide an overview of the size distribution exhaled for a variety of human activities from breathing to coughing. Results clearly show that humans emit many particles that are aerosolized in different size modes associated with distinct processes arising from deeper or less deep in the respiratory tract.

Another physical parameter of exhaled air is its temperature which is lower than the human body temperature but can still be much higher than the ambient temperature, especially in wintertime at mid latitudes. Values of around 32–34 °C have been widely reported (Carpagnano et al., 2017). Therefore, emitted puffs of air can rise by buoyancy in colder air. The effect of buoyancy and the impact of the exhaled temperature on the behavior of exhaled puffs and the microdroplets they contain have been discussed by Bourouiba et al. (2014) in their paper about expiratory events.

In addition to these physical aspects, it is also important to characterize the emitted aerosol from a biological point of view. The coronavirus content in the fluid of respiratory tracts of infected people has been studied (To et al., 2020; Zou et al., 2020). However, such studies do not allow a quantitative estimation of aerosol infectivity to be deduced. One of the most used models in aerosol contamination is that of Wells-Riley which uses the concept of a quantum of infection and is described in the next section. The quantum of infection rate of production per infected person is subsequently determined by epidemiological observations. A more recent concept is the Minimum Infective Dose (hereafter MID) which can be defined as the minimum dose of viruses that can initiate infection in a given proportion of receivers. The factors influencing this dose are important for the development of any risk assessment. MID estimates are often determined by infecting young, healthy volunteers, which is of course restricted to non-dangerous viruses such as those responsible for the common cold (Yezli and Otter, 2011). The value of the MID is influenced by a variety of factors such as the route of inoculation, vulnerability of volunteers etc. Therefore, the links between the MID and the quantum of infection is not straightforward (Jones and Su, 2015; Sze To and Chao, 2010; Yezli and Otter, 2011). Although details of these biological and medical characteristics of disease transmission are beyond the field of competences of the present authors, what can be retained is that it is widely recognized that the virus dose received by a receiver is the main parameter of disease transmission. This justifies the quantification of relative risk using concepts based on level of exposure which are developed in section 3, as long as comparable situations are considered.

2.2. Behavior of the emitted aerosol

Many of the recommendations of the WHO and government agencies have been based on an analysis of the dynamical behavior of a single particle in still air. However, an aerosol is in fact a two-phase medium (gas plus particles) with a much more complicated behavior, as is well known in the physics of atmospheric pollution. Several researchers have therefore been interested in the description of the air flow emitted by a person, either as gas (Gupta et al., 2010) or as aerosols (Bourouiba et al., 2014; Gupta et al., 2010).

Another point often not considered in simple analyses, although developed very early by Wells (1934), is the evaporation of exhaled microdroplets. Depending on the temperature and relative humidity, microdroplets can rapidly vaporize and experience a loss of more than 50% of their initial size, nearly an order of magnitude in mass. This leads to the formation of very infective "dry nuclei" (Nicas et al., 2005) which remain in aerosol form with a much higher viral load than the original droplets. However, the seminal work of Wells concerning evaporation, suffers from some simplifications and has been revisited. For example, Xie et al. (2007), reworked these calculations (70 years later) in a more precise fashion, taking the relative humidity into account, the residence time in the puff of moist air breathed out and the speed of the droplets in the atmosphere (increase in the Nusselt number). The results are qualitatively the same, but they find that the limiting size of the "large" droplets (falling to the ground before evaporation) is lower. Thus, the limiting diameter of the large droplets varies from 60 μm to 120 μm when the relative humidity goes from 90% to 0%, while Wells found from 97 μm to 172 μm. The limiting distance over which the large droplets fall goes from 1 m (1 ms^{-1} respiration speed) to 2 m (cough at 10 ms^{-1}) or 6 m (sneezing at 50 ms^{-1}).

In the same way Chong et al. (2021) presents a numerical simulation for the cough. The results are quite comparable. The "small" droplets are entrained by the turbulences of the puff and do not fall, hence producing airborne contamination.

Qualitatively, these results indeed show that the lifetime (relative to evaporation) of the droplets expelled, increases when the ambient relative humidity is greater, which is the case in an emitted puff. It means, as discussed above, that the larger droplets could have the opportunity to fall to the ground before reduction of their size by

evaporation. However, as soon as the droplets leave the puff, they evaporate more rapidly and stay longer in suspension either as smaller droplets or dry nuclei, thus increasing the risk of airborne transmission. In all cases, the "small" droplets are entrained in the puff and then remain in suspension due to their negligible falling speed.

In the present paper, performing precise calculations such as those of Xie or Chong concerning droplet evaporation, is not necessary since what is considered is a simple assessment of the relative risk for airborne transmission between outdoor and indoor situations, assuming many factors such as the distribution of emitted droplets and their behavior in a puff as being equal in both situations. Simple aspects of droplet vaporization and of the emitted puffs are discussed mathematically in the SM.

Based on the work of Bourouiba (Bourouiba et al., 2014) and others (Drossinos and Stilianakis, 2020), it can be seen than the recommended social distancing of 1–2 m is much too low, the puffs emitted by an infected people being able to travel over much larger distances and more particularly to rise due to buoyancy and be sucked into the air intakes of HVAC (Heating Ventilation Air Conditioning) systems, in case of indoor contamination.

Examination of the literature then shows that indoor HVAC systems are generally prone to homogenize indoor aerosols which justify the use of well-mixed models as in the present paper.

From the biological point of view, viruses can be inactivated (*i.e.* lose their infective power) either in aerosol form or on surfaces with a characteristic time (often called the lifetime) which depends strongly of physical parameters such as temperature, humidity or UV radiation field (Ijaz et al., 1985; Leclercq et al., 2014; van Doremalen et al., 2020). Assuming an exponential decrease of virus infectivity with time, introducing it into our calculation is easy, but, based on present knowledge of lifetimes, does not alter the main conclusions on the assessment of the relative risk between outdoors and indoors. Then for the sake of simplicity of the presentation, this point is discussed only in the SM.

3. Modeling indoor transmission of disease

3.1. Exposure level and disease probability

For harmful airborne substances whether chemical (gases), physical (asbestos, soot) or biological (virus, bacteria), it is possible to distinguish between a level of exposure and the probability of developing an illness or even dying, (especially in the case of poisonous gases). The level of exposure is often given as a concentration, either in mass or molecules per unit volume, since, multiplied by the pulmonary respiration rate and the time of exposure, it yields a dose which is clearly the risk factor for contracting an illness (for example cancer from asbestos). Note that if a number of molecules per unit volume is considered, then the dose is without dimension which is extremely useful in order to define the probability of developing a disease. This probability must be a strictly increasing function of the dose varying from zero (no exposure) to one (certainty of developing the disease above a given exposure). Most often employment legislation regulates the level of exposure in order to minimize health risk *i.e.* the probability of induced disease.

There have been a large number of attempts to model the transmission of respiratory diseases, most of them being related to indoor situations. The most famous is the Wells-Riley model and its various avatars (Ai and Melikov, 2018; Riley et al., 1978; Stephens, 2013) which will be described in the next section. Models use factors identical to the level of exposure, such as quantum of infection or Minimum Infective Dose and develop links to probability or percentage of infection. The quantum of infection covers the large variety of physical and biological processes involved in infection but for the purposes of the present paper, we shall just consider inhalation into the respiratory tract of micro-droplets produced by other humans. Thus, in this work, the level of exposure to viruses is considered as proportional to the Inhaled Flow Rate of Exhaled Particles, hereafter designed by as IFREP, which is the inhaled flow rate of particles which have already been exhaled by others (including healthy and infected people). We define the Inhaled Dose of Exhaled Particles (IDEP) as the product of IFREP by the time of exposure Δt. Of course, for disease transmission, the proportion of infected people needs to be considered but for comparable situations, the relative level of exposure between indoor and outdoor situations is then the ratio of the respectively calculated IFREP. The time of exposure can readily be considered through IDEP. Note that a similar approach has been developed by other authors for the indoor case only (Issarow et al., 2015; Rudnick and Milton, 2003).

By comparable situations, we mean the same population distribution with the same relative number of infected people. The present paper does not compare special indoor environments such as healthcare facilities, especially COVID units, with general outdoor environments. On the other hand, it is perfectly relevant to compare for example, an open outdoor market with a closed indoor supermarket.

Reducing the risk can be achieved clearly by minimizing the level of exposure but knowledge of the probability of infection requires developing a relationship with this exposure level. It will be shown later in this paper that for a Poisson probability law, it is easy to link relative probability of infection to the relative level of exposures.

3.2. The Wells-Riley model

Following his visionary intuition (Wells, 1934) that respiratory diseases can be due to exhaled airborne particles, Wells developed a model of airborne transmission for tuberculosis (and later other respiratory diseases) known as the Wells-Riley model and widely used up to the present day (Ai and Melikov, 2018; Riley et al., 1978; Stephens, 2013). Riley was a student and later on a collaborator of Wells. An excellent historical review of their findings and model development can be found in the Master's thesis of Johnstone-Robertson (2012). Recognizing that the amount of emitted human aerosols from people known as "infectors" was the equivalent to an exposure level, Wells introduced a quantity that he named the "quantum of infection" proportional to a number of infective airborne particles. The very mechanism by which infective particles trigger a respiratory disease is far from being fully understood, even today. It involves a variety of processes such as the deposition of particles in the respiratory track, (Nardell, 2016; Sze To and Chao, 2010). The great advantage of this notion of quantum of infection is that it clearly incorporates this variety of processes without seeking to establish mechanisms. Wells (Riley et al., 1978; Wells, 1955) introduced a quantity q which is a rate of production of quantum per unit time, per infected person (infector). The equivalent to the dose of exposure, in the notation of Riley (1978), can then be defined as $I \times q \times p \times t/Q$ where I, p, t and Q are respectively the number of infectors, pulmonary ventilation rate (volume/unit time), time of exposure and the rate of room ventilation with fresh air. The above quantity is a number without dimension and is calculated for a stationary state.

As discussed in the previous subsection, exposure and dose level are not the probability to develop a disease. Therefore, Wells and Riley introduced a probability of infection P following a Poisson law:

$$P = 1 - \exp(-I \times q \times p \times t / Q) \tag{2}$$

Note that except for the quantum of infection rate q and the number of infectors I, other quantities in this equation are well known for any disease. Hence, the quantum of infection production rate q per infector needs to be determined by epidemiological studies in situations where the number of infectors and infected can be estimated. For new emerging viruses, this quantity are in general unknown at the onset of the epidemic and therefore, need to be determined in order to make forecasts regarding the spread of the disease.

The Wells-Riley model refers to an indoor situation and assumes perfectly mixed air. This is normally the case for HVAC systems where most of the indoor air is recirculated and used as the heat transfer fluid. Fresh air is of course introduced in order to have a reasonable air quality

and to remove possible pollutants. There are regulations which set the norm for fresh air volume renewal q_{norm} per unit time and per person, depending on the situations. They have been set considering ordinary pollutants. Therefore, ventilation is also needed in these situations where no heating or cooling is required or where heating is done by radiant sources. There are in fact a variety of indoor ventilation schemes including natural ventilation, displacement ventilation, mixing ventilation and underfloor air distribution (Ren et al., 2016). For example, displacement ventilation is used to create a stratification of the air in a room and to have a lower concentration of pollutant at the height of a person (see (Bhagat et al., 2020)) than close to the ceiling. Therefore, the Wells-Riley model cannot be applied in this case or would need some adjustments. However, as said above, mixed ventilation is most common for heating and air conditioning systems.

3.3. A simple homogeneous model of IFREP and IDEP

In this sub-section, a simple calculation of the IFREP in an indoor space of volume V (area A, height h) is presented. The situation is depicted in Fig. 1.

Let N_p be the number of people inside, $N_i(t)$ the total number of aerosol particles of human respiratory origin inside the volume, it being assumed that recirculation, which is present in most HVAC systems, or by the movements of people, ensures an homogeneous mixing of the particles resulting in a concentration of particles of $n_i(t) = N_i(t)/V$. No consideration for the distribution of particle size is given here. For simplicity we shall take this as mono-disperse. The infective power either as "quantum of infection" or MID is not considered either. Again, this is justified by the fact that the purpose of our calculation is just to compare IFREP and IDEP in comparable indoor and outdoor situations. The mean exhaled flow rate of a person is taken as q_1 (of course identical to the inhaled rate) and the concentration of particles in this flow will be assumed equal to n_1. The flow rate of fresh air introduced in the volume is q_2. A typical value for q_2 can be taken from HVAC standards (Legg, 2017; Lemaître, 2011) which ideally give the renewal flow rate per individual:

$$q_2 = N_p \times q_{norm} \tag{3}$$

Typical values of q_{norm} range from 20 to 60 m^3/h/person.

The mean air exhaust flow rate of the building is equal to the mean fresh air inlet flow rate q_2 considering the characteristic time of the problem. As in the Wells-Riley model, we do not consider the possible variation of this flow rate with time.

The differential equation which governs the temporal evolution of N_i can then be written:

$$\frac{dN_i}{dt} = V \times \frac{dn_i}{dt} = N_p \times q_1 \times n_1 - q_2 \times n_i \tag{4}$$

Assuming no exhaled particles at zero time, the solution for n_i is straightforward:

$$n_i(t) = n_i^{\infty} \times \left[1 - \exp\left(-\frac{t}{\tau_1}\right)\right] \tag{5}$$

with:

$$n_i^{\infty} = \frac{N_p \times q_1 \times n_1}{q_2} \tag{6}$$

and:

$$\tau_1 = \frac{V}{q_2} \tag{7}$$

Considering (eq. (3)), it is seen that the value of n_i for $t \gg \tau_1$ *i.e.* n_i^{∞} is a function of the norm (inlet fresh air/person) and human respiratory characteristics and not of the number of people:

$$n_i^{\infty} = \frac{q_1 \times n_1}{q_{norm}} \tag{8}$$

As in the Wells-Riley model this quantity refers to a stationary state. For a person arriving in the building at time $t \gg \tau_1$, the value of IFREP will be $IFREP_{indoor} = n_i^{\infty} \times q_1$ and if they stay there for an interval of time Δt, then the value of IDEP will be $IDEP_{indoor} = IFREP_{indoor} \times \Delta t$.

This simple model can be easily extended to the nonstationary case as discussed in the SM. For indoor situations, it is quite similar to the so-called "rebreathed air" models (Issarow et al., 2015; Rudnick and Milton, 2003).

3.4. Link between $IDEP_{indoor}$ and the Wells-Riley model

The model presented in section 3.3 can be compared to the Wells-Riley model where a quantity without dimension that can be considered as a dose is defined:

$$X = \frac{I \times q \times p \times t}{Q} \tag{9}$$

The various quantities in this equation have been defined in section 3.2. From section 3.3 it is easy to show that the quantity that we have defined as $IDEP_{indoor}$ can be written as:

$$IDEP_{indoor} = \frac{(N_p \times q_1 \times n_1) \times q_1 \times \Delta t}{q_2} \tag{10}$$

It is clear that the quantities q_1, q_2, and Δt are exactly the same as the respective quantities p, Q, and t defined by Wells-Riley. However, the formulation of $IDEP_{indoor}$ does not take into account a number of infectors and a quantum production rate. The reason is that this is unnecessary for estimating a relative risk (target of the present paper) between two situations (indoor and outdoor) where the proportion of infectors and their production of infective particles are assumed to be the same. It is straightforward however, to deduce from the above equations that:

$$I \times q = \alpha \times N_p \times q_1 \times n_1 \tag{11}$$

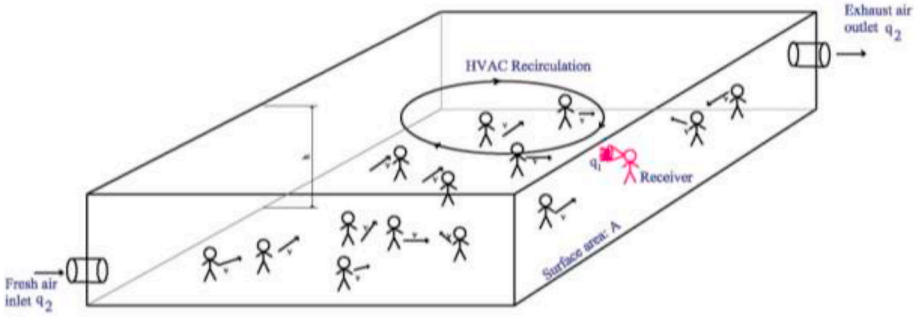

Fig. 1. A schematic description of a typical indoor situation.

where α is a proportionality factor. Further, it seems rational to assume that the number of infectors is proportional to the total number of persons in the building:

$$I = \beta \times N_p \tag{12}$$

from where it follows that the quantum of infection could be expressed in the form:

$$q = \gamma \times q_1 \times n_1 \tag{13}$$

β and γ are proportionality factors and $\alpha = \beta \times \gamma$.

Note that this approach, although not expressing the value of the proportionality factors, is very similar in its principle to the estimation of quanta emission rates of SARS-CoV-2 developed by Buonanno et al. (2020).

3.5. Inhomogeneous models

The Wells-Riley model is based on two main assumptions: the indoor air that is inhaled by the human receptor is well mixed and in a steady state. The indoor IFREP model developed above is easy to use for the transient state where the concentration of infective particles (or quantum) is increasing as discussed in the SM.

Evaluating the airborne infection risk considering spatial resolution is much more challenging as discussed by Zhang and Lin (Zhang and Lin, 2021 and references therein). Some attempts in this direction have been made and can involve Computational Fluid Dynamics (Li et al., 2018; Vuorinen et al., 2020; Zhang and Lin, 2021). However, in essence inhomogeneous models are devoted to particular situations and drawing general conclusions on the relative outdoor versus indoor case seems beyond their possible field of applications.

4. Outdoor transmission: A simple airshed model of IFREP

We take now the situation depicted in Fig. 1 and remove the walls and ceiling to imagine the same situation transposed "outdoors". This is depicted in Fig. 2. We use here what is called an "airshed concept" which is used in the analysis of city air pollution (Cushman-Roisin, 2012). The problem is analyzed from the perspective of a material balance over a specific part of the atmosphere, the airshed. Although this volume cannot be accurately defined, with the same precision as water in a pool, it is a useful concept. Its definition is of course strongly dependent on atmospheric conditions.

The surface of area A has a width along the wind l and a length across it, L. The wind velocity is V_∞. Other parameters have the same meaning as in section 3.3 and Fig. 1, and $n_i(l)$ is the concentration of human exhaled particles at the downwind limit of area A.

For typical situations, with l comprised between say 20 m and 1 km the hydrodynamic time scale $t_h = l/V_\infty$ for most usual wind conditions, is from a few seconds to a few minutes and mostly well below 1 h. Assuming a stationary state and that most of the respiratory human particles are well mixed and will exit the surface area downwind below a typical height H, the following conservation equation can be written:

$$n_i(l) \times L \times H \times V_\infty = q_1 \times n_1 \times N_p - loss \tag{14}$$

The term "*loss*" takes care of possible particle losses above the height H together with lateral ones. Setting this term to zero in fact maximizes the evaluation of the outdoor risk. Then $n_i(l)$ can then be written as:

$$n_i(l) = \frac{q_1 \times n_1 \times N_p}{L \times H \times V_\infty} \tag{15}$$

and $IFREP_{outdoor}$ for an outdoor receiver downwind of the surface A will be: $IFREP_{outdoor} = n_i(l) \times q_1$.

Note that minimizing the value of H in equation (15) also maximizes directly $IFREP_{outdoor}$ and therefore the level of exposure which can be a deliberate choice, as discussed below.

Note that eq. (15) can be written as:

$$n_i(l) = \frac{q_1 \times n_1 \times D_p}{V_\infty} \times \frac{l}{H} \tag{16}$$

where $D_p = N_p/(l \times L)$ is the number density per surface unit of persons outdoors.

Determining the quantity $H = f(l)$ is an extremely complicated problem of atmospheric physics. H can be defined as a height above which the total flow rate of human exhaled particles becomes negligible for the airshed balance *i.e.* much lower than its counterpart from ground to this same height. But the choice can also be made to deliberately take a lower value if it leads anyway to a risk much higher than the real outdoor one. Deriving its value from scratch and only basic principles is a virtually impossible task. However, its order of magnitude can be evaluated from a large number of studies in the field of atmospheric pollution, based on theory as well as on experimental observations. Before presenting this evaluation, simple analytical expressions are derived in the next section for the relative level of exposure and the relative probability of infection.

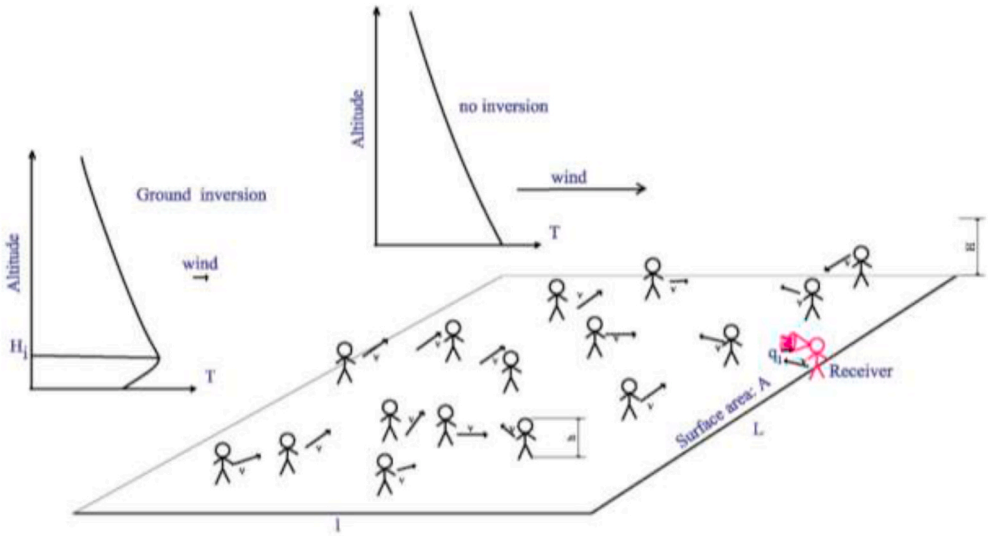

Fig. 2. Same situation as in Fig. 1 but outdoors with two possible meteorological situations depicted.

5. Comparison indoors versus outdoors

5.1. Simple assessment of relative level of exposure indoors versus outdoors

The purpose of this paper is essentially to assess the relative aerosol contamination risk between outdoor and indoor situations. As stated above, we assume two comparable situations *i.e.* the proportion of infected people is the same with the same characteristics (exhaled flow rate, particle concentration and viral load within the flow) and that "receivers" present the same sensitivity to infection. The relative risk can be evaluated from the quantity that we have defined and evaluated (under a simple hypothesis) in the previous sections as IFREP and IDEP. Note that IDEP which is the product of IFREP by the exposure time Δt, is the real factor of risk. Both quantities consider only the continuous inhalation of the bulk air. Crossing (or contact) transmission which can occur when you cross a person but also if you stay "downwind" of this person for a given time can be treated only by inhomogeneous models and is not considered here. This is true for both indoor and outdoor situations.

Then for the same exposure time, the relative level of exposure R between outdoors and indoors can be estimated as the ratio $IFREP_{outdoor}/IFREP_{indoor}$ which can then be written as:

$$R = \frac{q_{norm}}{V_\infty} \times D_p \times \frac{l}{H} \tag{17}$$

Note that q_1 and n_1 vanish. In this form, the only quantity which refers to the indoor situation is q_{norm}. This is due to the fact that the ventilation rate of fresh air is normally proportional to the number of people indoors. D_p is the number density of persons outdoors (person/square meter), $D_p = N_p/A$, the quantity H/l will be discussed at length in the SM where it will be shown that it is linked to the meteorological conditions and to the wind V_∞ itself.

Indeed, a correlation between the epidemic and wind, as well as atmospheric conditions, (including pollution) which yield the l/H factor, has been observed (Al-Rousan and Al-Najjar, 2020; Rendana, 2020). Correlation is not causality, but the above formula sheds clear light on the observations.

Note that a different formula can be derived for situations where the indoor ventilation rate does not follow the norm but for the sake of conciseness and clarity, this development can be found in the SM.

5.2. Probability of infection

Like others, including Wells and Riley (Riley et al., 1978; Wells, 1955), we use a Poisson law of probability to compare the infection probability indoors and outdoors (P_{indoor} and $P_{outdoor}$ respectively) for similar situations.

$$P_{indoor} = 1 - \exp(-X_{indoor}) \tag{18}$$

The value of X_{indoor} in this model has been presented previously and is in fact proportional to IDEP (and therefore IFREP). As detailed in section 3.4, it is possible to make a parallel between the quantity that we use in our IFREP calculation and the X used in the Wells-Riley model.

The calculations and concepts presented in previous sections allow us to calculate a relative level of exposure between outdoors and indoors which can translate directly into a relative value of X:

$$X_{outdoor} = R \times X_{indoor} \tag{19}$$

From here, we can derive that, for comparable situations, the outdoor probability of non-infection is linked to the indoor one by:

$$1 - P_{outdoor} = (1 - P_{indoor})^R \tag{20}$$

where 1-P is the probability of not being infected. The factor R, if $\ll 1$, leads to a tremendous advantage of the outdoors in many situations.

It has to be noticed that for small values of X, and therefore of P, respective Taylor expansions of the probability show that the ratio of probabilities of being infected reduces to the R factor.

5.3. Atmospheric conditions, dispersion and choice of H/l

The choice of H/l is clearly central in the assessment of relative level of exposure and probability of infection between indoor and outdoor situations. Indoor modeling of a well-mixed homogeneous situation is governed by simple considerations, assuming complete stirring by convection currents in a closed finite volume. Diffusion therefore, does not need to be considered. The outdoor airshed balance is also based on simple considerations but as stated above, includes an unknown quantity *i.e.* the height of the airshed. The determination, even approximative, of its value obeys different phenomena, generally much more complicated and harder to consider than in the indoor situation. Firstly, the advection by the wind, which is always present, even be it very weak and not constant, in intensity as in direction. Besides this transport by advection, atmospheric turbulence contributes to the dispersion by so-called eddy diffusion (much more efficient than molecular diffusion). This important point is related to the stability of the atmosphere (see SM), which in turn depends on meteorological conditions, solar exposure, and thermal radiation from the ground and from the clouds.

H could have been taken as the so-called mixing height (Holzworth, 1974) which can be defined as the vertical height over which an unstable parcel of air taking off from the ground will rise (see below). Therefore, it corresponds in fact, to the maximum height of the atmosphere where mixing of pollutant occurs. This quantity can change from zero in situations of inversion to thousands of meters (Holzworth, 1974). Therefore, it seems more reasonable for the present case to use a model of the dispersion of gaseous pollutants on a more reasonable length scale.

There is a large literature on the plumes and puffs emitted from smoke stacks and the estimation of pollutant concentration downwind due to dispersion, see for example the excellent course (Pilat, 2009) and the review by Holmes and Morawska (2006). There are also numerous papers linked to biology which treat odor dispersion, for example of insect pheromone downwind (Farrell et al., 2002). Our problem can be thought of as a field with several analogs to a small smokestack producing airborne particles, the human emitters. We make use of what is known of the vertical length of dispersion, extremely dependent of meteorological conditions, to evaluate H.

For the sole purpose of lightning the text of the present article, we refer the reader to the SM for further reading on atmospheric conditions and stability and their influence on the dispersion of plumes and puffs. In this document and following Pasquill (1961), classes of atmospheric stability are defined ranging from A (very unstable) to F (very stable), D referring to a neutral atmosphere (as defined in the SM). In the case of a Gaussian vertical dispersion, it is shown that for typical lengths involved in many practical situations (an outdoor market for example) H is proportional to l which implies the choice of a constant value for H/l shown in Table 1.

However, not all the plumes can be considered as being described by a vertical Gaussian dispersion. In some meteorological situations there are hardly any vertical dispersions (situation corresponding to "fanning" and "fumigation", described in SM). In this very special case, which corresponds to a very stable and strong inversion situation, we assume a constant value for H that will be taken as the mean height of an emission source *i.e.* 1.5 m.

Table 1
Adopted values of H/l for vertical Gaussian dispersion and various meteorological conditions (see SM).

	A	B	C	D	E	F
H/l	0.2	0.097	0.074	0.051	0.034	0.023

5.4. Numerical application and discussion

In the present section, we assume that the indoor ventilation rate is fixed accordingly to the norm. In Table 2, some values of factor R obtained for a few typical meteorological conditions and outdoor person densities, are listed for the case of a vertical Gaussian dispersion.

The first one is the situation of a very unstable atmosphere which corresponds to heated ground and can be found in the daytime in summer at mid-latitudes or in the dry season in a tropical area. Another situation is a stable but moderately windy condition, corresponding for example, to a winter day at mid-latitudes. The last one corresponds to a very stable atmosphere with low wind. In this table, for the evaluation of the relative risk factor R, defined in equation (17), we have taken a mean value of q_{norm} of 30 cubic meter/hour/person for the estimation of the indoor level of exposure. Note that the wind here is taken as its mean value at 10 m of altitude. In the real world, the wind has a vertical profile, starting from zero at the ground and increasing with altitude (Wikipedia, 2020), see also the discussion in the SM. Of course it is also a temporal mean (over 10 min) as the wind is always turbulent and variable.

In Table 3, results are presented for a constant height of dispersion (1.5 m) versus downwind distance to emitters. This corresponds to special and very stable meteorological conditions as discussed in the previous section.

The main conclusion that can be drawn from Tables 2 and 3 is that, most generally, outdoor risk is much less than indoor (in most cases by orders of magnitude). Only situations of inversion with low wind and a very stable atmosphere, which are prone to atmospheric pollution, could promote an outdoor transmission close to that indoors, especially in crowded areas. If this situation occurs the "fresh" air that is introduced from outside in buildings could already be "polluted" leading to a strongly enhanced indoor airborne transmission of the disease. In the authors' opinion, this could explain some observed outbreaks of the epidemic depending on topography and meteorology as shown in a recent paper (Rohrer et al., 2020). Note the importance of the wind factor and of the outdoor person density.

6. Some markers of risk outside and inside

6.1. Anthropogenic aerosols outdoors

A correlation has been observed especially in Italy (Rohrer et al., 2020; Zoran et al., 2020) between pollution by particulate matter and the outbreak of the epidemic. It has been suggested that in a synergistic effect, pollution can increase the infective power of the virus by the agglomeration of airborne infective particles with PM particles. It is interesting to note that after the discovery by Robert Koch in 1882 of the mycobacterium responsible of tuberculosis, it was first thought that tuberculosis was spread by the breathing in of dust particles contaminated with dried mycobacterium tuberculosis laden sputum (Johnstone-Robertson, 2012) an idea which, was revealed to be wrong. As discussed by Doussin (2020) and in the SM, the typical characteristic time of agglomeration in the atmosphere is far too long to allow such an agglomeration. It is known that bacteria and viruses can be found in atmospheric aerosols (Kalisa et al., 2019), but most natural aerosols are formed by ground surface abrasion by the wind which explain the observation. If the ground is contaminated, then it could of course be the case for particles formed by abrasion.

Correlation is not causality but the observation of a peak of pollution due to particulate matter is clearly linked with special meteorological conditions prone to enhance this pollution. Following the present work, we suggest that the cause of **the outbreak of epidemics, is these meteorological conditions and not the particulate matter pollution itself**. As meteorology is predictable a few days in advance, this could be used for public recommendations and alerts. Note that it is well known that topography can strongly influence pollution by particulate matter. An example is perfectly described in p 204, of (Cushman-Roisin, 2012) in the case of the city of Los Angeles, prone to this kind of situation since it is cornered between on the one side, an arc of mountains and ridges and the Pacific Ocean on the other side.

Table 2
Relative risk factor R (outdoor/indoor) for Gaussian vertical dispersion, various atmospheric conditions and people density.

			R factor		
Atmospheric stability	H/l	wind (m/s)	$D_p = 0.25$	$D_p = 1$	$D_p = 3$
very unstable A	0.2	1	0.010	0.042	0.125
very unstable A	0.2	3	0.003	0.014	0.042
neutral D	0.051	2	0.020	0.082	0.245
neutral D	0.051	6	0.007	0.027	0.082
very stable F	0.023	0.5	0.181	0.725	2.174
very stable F	0.023	2	0.045	0.181	0.543

Table 3
Relative risk factor R (outdoor/indoor) in strong inversion and low wind conditions for various lengths along the wind l and people density.

		R factor		
wind (m/s)	l (m)	$D_p = 0.25$	$D_p = 1$	$D_p = 3$
0.5	50	0.14	0.56	1.67
0.5	100	0.28	1.11	3.33
2	50	0.03	0.14	0.42
2	100	0.07	0.28	0.83
2	200	0.14	0.56	1.67

6.2. Carbon dioxide indoor

In air exhaled by humans, carbon dioxide has a much larger concentration (4–5%) than in fresh outdoor air (section 2.1). Therefore several authors (Rudnick and Milton, 2003) have proposed to monitor CO_2 levels in indoor situation as an indicator of the risk of infection. In fact the simple model developed for IFREP in section 3.3 can be readily applied to indoor CO_2. Noting $[CO_2]$ the concentration of CO_2 in ambient air it leads to:

$$[CO_2] = [CO_2]_0 + ([CO_2]_\infty - [CO_2]_0) \times \left[1 - \exp\left(-\frac{t}{\tau_1}\right)\right] \tag{21}$$

where $[CO_2]_0$ is the initial concentration of CO_2 in fresh air and $[CO_2]_\infty$ the concentration at stationary state which can be written as:

$$[CO_2]_\infty = [CO_2]_0 + \frac{q_1 \times [CO_2]_{exhaled}}{q_{norm}} \tag{22}$$

where $[CO_2]_{exhaled}$ is the concentration of CO_2 in the exhaled air.

The characteristic time is the same as for IFREP hence the interest of monitoring CO_2. With typical values of respiratory parameters and q_{norm}, it is found that in a stationary state, the amount of CO_2 in air is easily twice that in fresh air, making monitoring easy. An increase of q_{norm} by an order of magnitude yields only an increase of around 10% of the concentration in a stationary state, therefore monitoring would still be possible with accurate sensors.

7. Conclusions and recommendations

The last months have seen an extraordinary inflation of papers dealing with the COVID-19 and its transmission. A great number of these papers deal with correlation and observation and not with quantitative models of the physical processes of transmission. The fact that correlations are not causalities makes their use difficult for public decisions to mitigate virus spread preserving social life and the economy as much as possible. For example, it is clear that a measure to reduce pollution by

particulate matter emitted by combustion (as taken for transportation in urban areas), will have no effect on the spreading of the disease in view of the arguments developed in section 6.1 and in the SM.

Airborne transmission of COVID-19 is now widely recognized, and this has led public authorities to recommend or impose the wearing of mask in the general population, certainly an excellent mitigation measure as shown by observation. However, when it comes to the question to know when to wear it, the answer is far from being so obvious, although it is clear that wearing it night and day and in all circumstances is not realistic.

In this paper, founded on quite simple calculations, we have presented a quantitative assessment of the relative risk of airborne virus transmission for the outdoor versus indoor situations. Calculations result in remarkably simple formulas which, considering the science of atmospheric physics and pollution, allows us to assess the relative risk between indoor and outdoor situation. The simplicity of this derivation could be criticized, and its application is most often based on proportionality rule. However, the beauty of these simple formulas makes sense.

First and as discussed in section 5.4, it shows that even in crowded areas, the risk outdoors is much less than indoors. From this point of view, it has to be noted that some decisions taken by public authorities could have appeared as absurd to the man in the street. This opinion just based on common sense is confirmed by the present study. Examples are very numerous ranging from the lockdown of open markets when indoor supermarkets were open to the public, or prohibition of a variety of outdoor sport and exercise. Let us point out that such lockdowns and prohibitions can have profound impact on the health of citizen and economy. Note that in the present paper, we have not considered the phenomena of droplet evaporation indoors which is strongly enhanced by the low relative humidity due to heating in wintertime, and clearly will lead to an even higher indoor risk, reinforcing our conclusion.

Considering mitigation measures, it is clear that wearing a mask, especially indoors, is an important way to reduce the risk of contamination (whether from contact or from airborne). However, and as noted by Morawska et al. (2020), other measures should include strongly increasing ventilation which means increasing the fresh air renewal norm per person by an order of magnitude either in HVAC systems or by natural ventilation (Escombe et al., 2007). If this is not possible, apparatuses allowing indoor air to be sterilized, should be envisaged, however, to be efficient, here again they would need to be able to treat a flow rate at least an order of magnitude higher than the present norm.

Following our study, we are led to believe that the fact that Africa, especially sub-Saharan, has not been stricken by the disease as much as rich mid-latitude countries, is linked to climatic factors (very unstable atmosphere) together with an outdoor way of life (for example outdoor markets instead of air-conditioned indoor supermarkets). Due to the low gross national product per inhabitant, the use of air-conditioning is also much less common than in richer countries of the same latitude. On the other hand, a situation of temperature inversion can occur within the day, especially in wintertime for mid-latitudes where the sun is low in the sky and supplies less warmth to the Earth's surface. Either radiation or the so-called subsidence inversion (*i.e.* above the ground) can inhibit vertical dispersion and act as a lid and trap cold air at the ground. These effects can be strongly amplified in mountain valleys. We suggest that, in these geographical areas prone to pollution by secondary PM 2.5 which include large urban areas with collective housing and apartment blocks, monitoring this pollution, together with meteorological forecasts, could be a way to alert the population of risky days and to reinforce mitigation measures for short periods of time. Such a suggestion has already been given for indoor CO_2 levels (Rudnick and Milton, 2003).

To finish we want to emphasize that the spreading of the disease is an extraordinarily complex phenomena certainly not restricted to the airborne transmission way, although this way can make the difference for the effective reproduction number leading or not to an epidemic burst. The modest contribution of the present paper is an attempt to quantitatively assess the relative risk linked to aerosol between outdoor and indoor situations. We hope that it will encourage atmospheric physicists and pollution experts to tackle the outdoor dispersion problem of respiratory produced aerosols in more detail.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

The authors wish to acknowledge Allan Crouvizier, Alexandre Modesto and Alexey Potapov for helpful discussions and bibliographic watch.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2021.111189.

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